

# Detector electrode introduced by mediastinoscopy for atrial triggered cardiac pacing

## *A follow-up study of electrode function in 82 patients*

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*An atrial detector electrode was introduced by mediastinoscopy in 82 patients requiring permanent cardiac pacing. There were no complications. An adequate P wave was recorded in 80 patients. During the following week, the P wave became ineffective in 5 patients; angina occurred in 2 and atrial arrhythmias in 2. Atrially triggered ventricular pacing was established in 73 patients and was followed in 71 patients for a period of 1 to 113 months. In 7 cases, it had to be terminated because of an ineffective or unstable P wave, in 6 cases because of atrial arrhythmias, and in 4 cases because of advanced age and recurrent infections.*

*The method is technically simple and places little stress on the patient.*

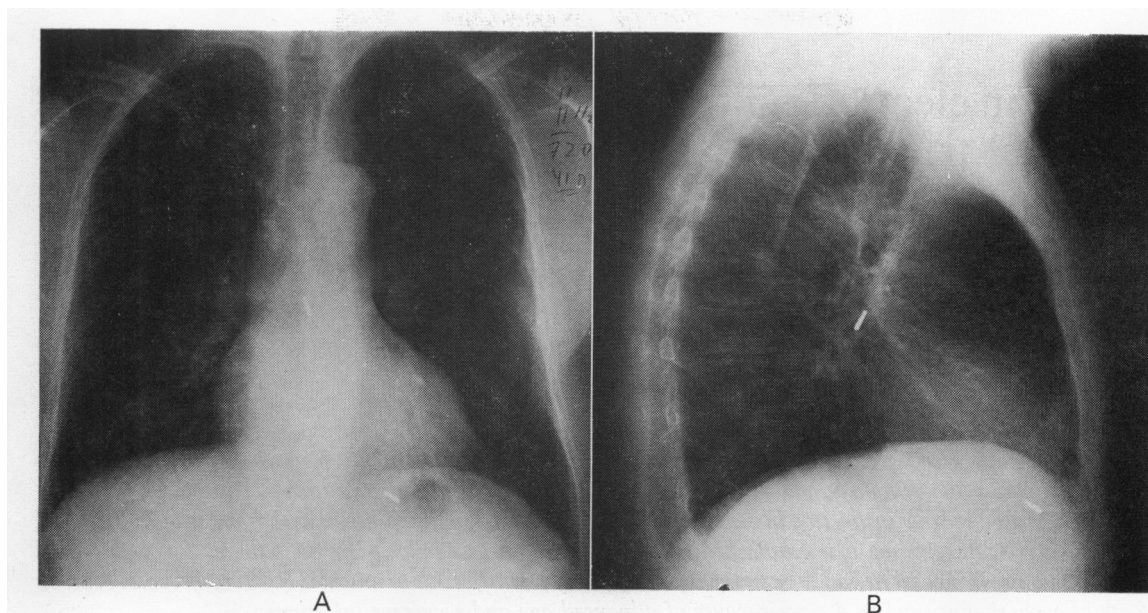
When permanent cardiac pacing was introduced (Weirich *et al.*, 1957; Furman and Schwedel, 1959; Glenn *et al.*, 1959; Senning, 1959; Elmqvist and Senning, 1960) the aim was to prevent Adams-Stokes attacks and death in patients with complete heart block. Fixed rate cardiac pacing meant an enormous benefit to these patients. With technical progress, greater demands have been made on cardiac pacing in order to avoid potentially dangerous interference between pacemaker rhythm and spontaneous beats and to help patients live a normal life without feeling discomfort or incapacity. Atrial triggered cardiac pacing means for many patients the achievement of this aim. The method was described in animal experiments by Folkman and Watkins (1957), Stephenson *et al.* (1959), Battye and Wheale (1960), and Nathan *et al.* (1963). It was introduced in the clinic by Center *et al.* (1963), who used an atrial sensing electrode introduced by thoracotomy.

At the Karolinska hospital atrial triggered cardiac pacing was introduced by Carlens *et al.* (1965) and Lagergren *et al.* (1966a, b) by means of an atrial sensing electrode introduced by mediastinoscopy (Carlens, 1959). The present paper reports a follow-up of operative results, the primary and late effectiveness of this type of atrial sensing electrode, and the cause of terminating atrial triggered cardiac pacing in all patients treated in this hospital by this method since 1964.

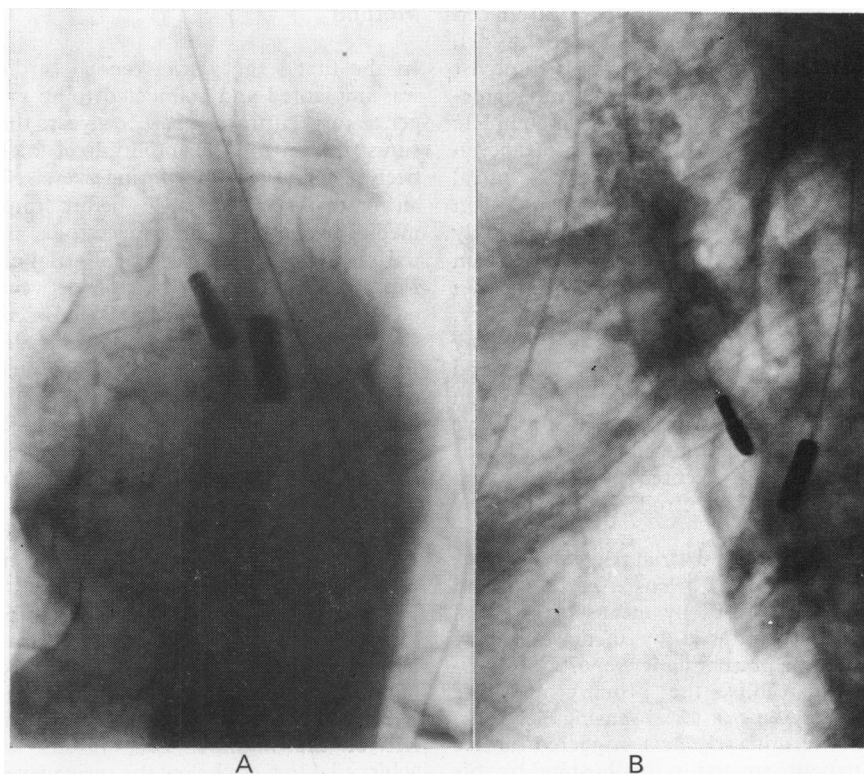
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### Method

In the first stage a transvenous cardiac electrode was implanted and connected to an external pulse generator. During the past few years the stability of sinus rhythm and the amplitude of the P wave have been tested at rest and during exercise by means of an oesophageal electrode before implanting the mediastinal atrial sensing electrode. Fig. 1 shows the position of the mediastinal atrial electrode and Fig. 2 shows the close relation between it and the oesophageal electrode. Fig. 3 shows the P waves simultaneously recorded from both electrodes and Fig. 4 the standard leads with the atrial triggered cardiac pacemaker. The electrode used for mediastinal implantation was the Siemens-Elema EMT 588, and implantation was performed under general anaesthesia. A small incision was made in the jugular fossa and the front of the trachea was dissected. Under visual control through the mediastinoscope the dissection was continued by a 3 mm thick closed end suction tube past the point of bifurcation of the trachea and behind the right branch of the pulmonary artery to reach the layer of the connective tissue between the posterior wall of the left atrium and the oesophagus. Attempts were made to make a narrow tunnel slightly to the left of the midline. Best results were usually achieved 4 to 5 cm below the carina level. When this region had been located the electrode tip was gently advanced and, with the aid of forceps, placed in the



**Fig. 1** X-ray film in anteroposterior (A) and lateral (B) views visualising the mediastinal and the ventricular electrode in adequate position.



**Fig. 2** X-ray in antero (A) and lateral (B) views showing the close relation between the mediastinal and the oesophageal electrode. The upper electrode belongs to the mediastinal atrial electrode.

connective tissue as close as possible to the atrial wall, while the amplitude of the P wave was checked on the electrocardiogram. Forcing the suction tube down involves the risk of both puncturing the pericardium and placing the electrode in an unstable position.

The amplitude from the positive to the negative deflection of the P wave was measured between the

mediastinal atrial electrode and the right arm. The amplitude was not significantly different if the reference electrode was placed in the region of the generator pocket in the abdominal wall. More recently the positive and negative deflection of the P wave was measured separately because the input sensitivity of the pulse generator is more closely related to the positive and the negative deflections of the P wave rather than to the total amplitude. In order to get comparable values the total amplitude is given on the figures.

During the first period of this study a Siemens-Elema EM 141 pulse generator was used, requiring a P wave amplitude of at least 0.9 mV. Later on the more sensitive pulse generator, Siemens-Elema EM 144, EM 156/50, was constructed, which was triggered by a P wave of 0.5 to 0.4 mV.

When the P wave reached a stable value of at least 1 to 1.5 mV a loop was made in the neck and the electrode cable was drawn stepwise subcutaneously by means of a long injection needle, over the chest and abdomen to the left groin. The long subcutaneous tunnel minimised the risk of infection. The procedure, which has undergone only minor variations since it was first introduced, places very little strain on the patient.

If a second intervention for correction of the electrode placement was undertaken, varying degrees of scar formation could be found anterior to the trachea. In such cases the soft tissue to the right of the trachea was dissected. At the level of the right main bronchus a narrow tunnel was then dissected by the suction tube to the left of the midline.

The amplitude of the P wave after the operation often decreased during the first week after implantation and was checked repeatedly during 10 days. Before the atrial triggered generator was implanted the patient was tested with an externally coupled atrial triggered pulse generator at rest and during exercise. The amplitude of the P wave was thereafter routinely measured when replacement of the pulse generator was made.

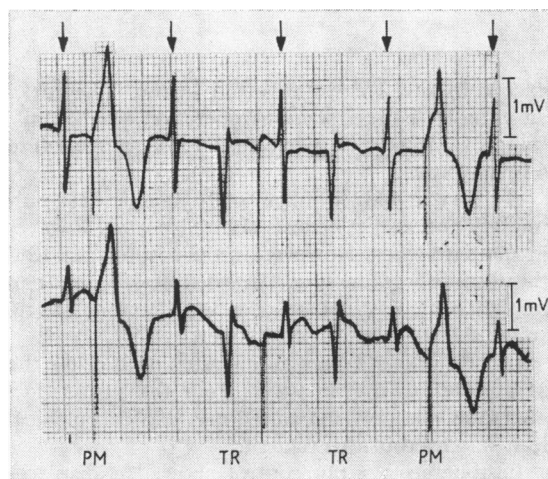


Fig. 3 Electrocardiogram from mediastinal atrial electrode and oesophageal electrode. Upper row, recording between the right arm and the mediastinal electrode. The mediastinal atrial electrode is, thus, not connected to the atrial triggered pulse generator which is stimulating at its basic rate: 52 beats per min. Lower row: recording between the right arm and an oesophageal electrode. The recording was made at the same time as the x-ray film shown in Fig. 2. P waves are indicated by arrows and resemble each other in the two leads. There are both transmitted QRS complexes (TR) and pacemaker induced QRS complexes (PM). The second pacer artefact arrives in the refractory period. Paper speed: 25 mm per second.

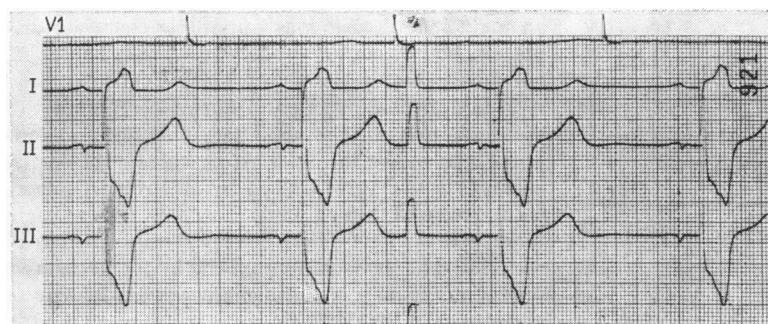


Fig. 4 Electrocardiogram with atrial triggered cardiac pacing. Standard leads with the mediastinal electrode connected to the atrial triggered pulse generator in the same patient as in Fig. 1, 2, and 3. The small negative deflection of the P wave in leads II and III is caused by the P wave triggering the pulse generator. Paper speed: 50 mm per second.

Table 1 *Review of case material*

No. of patients	Age Mean $\pm$ SD (range)	No. starting atrial triggered cardiac pacing	Duration of atrial triggered cardiac pacing No. mean $\pm$ SD months (range)	No. terminating atrial triggered cardiac pacing	No. of deaths	No. of patients still undergoing atrial triggered cardiac pacing
Men 44	55 $\pm$ 11 (26-70)	40	39* 39 $\pm$ 5 (1-103)	8	7	24
Women 38	52 $\pm$ 13 (29-69)	33	32* 46 $\pm$ 6 (4-113)	9	2	21

\*1 patient lost from follow-up.

## Subjects

The present study was based on 82 patients, 44 men and 38 women. The mean age was 55 years for men and 52 years for women (range 26 to 70 years) (Table 1). All patients in whom an attempt was made to introduce a mediastinal atrial pacing electrode in the period from 1964 to May 1974 were included in the study and all patients continuing to attend the Karolinska hospital for pulse generator replacements were included in the follow-up. In a few cases complementary information was obtained from hospitals outside the region of Stockholm.

In 9 patients atrial triggered cardiac pacing was not started (Table 2), and 2 patients with this type of pacing were lost from the follow-up.

The observation period in the remaining 71 patients ranged from 1 to 113 months. The mean observation period was  $39 \pm 5$  SD months for men and  $46 \pm 6$  SD months for women. During this control period 7 men and 2 women died. Death in 5 cases was not caused by pacemaker defect. In the remaining 4 cases the cause was unknown. In 17 cases (8 men and 9 women) atrial pacing had to be terminated. The reasons for this are discussed later. At the time of writing there are 45 cases (24 men and 21 women) still undergoing treatment.

## Results

### PRIMARY OPERATIVE RESULTS

An acceptable P wave with an amplitude of at least 1.0 to 1.5 mV was achieved in all patients

except in the 2 cases reported below. The mean amplitude of the P wave at operation was 2.8 mV (range 1.3 to 5.0) in men and 2.5 mV (range 1.0 to 6.0) in women.

A 38-year-old woman with congenital pulmonary and aortic stenosis showed normal P waves in standard leads. However, in the oesophageal lead the P wave was missing. An attempt was made to implant a mediastinal atrial pacing electrode but no suitable P wave could be obtained.

In a 68-year-old woman, operated on in 1964, the amplitude of the P wave from the mediastinal electrode was only 0.3 to 0.4 mV. No oesophageal lead was tried in this patient.

Postoperative x-ray control showed a displacement of the electrode into the pericardium in 2 patients but its position was easily adjusted: 1 of the 2 patients developed atrial fibrillation, which was converted, and when fibrillation subsequently recurred it was decided not to start atrial triggered cardiac pacing. In a third patient pleuropericarditis was observed 4 weeks after implantation; the exudate regressed spontaneously.

### AMPLITUDE OF P WAVE

During the first week after the implantation of mediastinal atrial pacing electrode there was often a decrease of about 1 to 2 mV in the amplitude of the P wave (Fig. 5). In 2 of the 78 patients this decrease caused an inefficient P wave. In another 4 patients an inefficient P wave was caused by electrode displacement visualised by x-ray film (Table 3). Measurements of the P wave after starting atrial triggered cardiac pacing and during replacement of the generator showed moderate variations in amplitude, as shown in Fig. 6. After starting the pacing an inefficient P wave caused by electrode displacement occurred in 6 patients (Table 3). In 3 of them there had obviously been a very strong traction of the electrode by rotation of the pulse generator, during pregnancy or during replacement of a pulse generator. In the other cases the cause was obscure.

Table 2 *Different causes of not starting atrial triggered cardiac pacing*

Ineffective P wave preoperatively	Ineffective P wave postoperatively	Anginal pains	Sinoatrial block	Atrial fibrillation
2	3	2	1	1

In one patient a broken electrode, which was easily repaired, caused an ineffective P wave.

#### CORRECTED AND RENEWED ELECTRODE IMPLANTATION

In 9 out of 13 patients with an inefficient P wave, attempts were made to correct or reimplant the mediastinal atrial electrode. In the other 4 patients already undergoing atrial triggered cardiac pacing this treatment was considered of less value for a variety of reasons, including advanced age. Out of the 4 cases in which an attempt was made to correct the mediastinal atrial electrode, one was successful one day after implantation and one successful one year after implantation. The two unsuccessful cases

had displaced electrodes before starting atrial triggered cardiac pacing. Reimplantation functioned well in 4 out of 5 patients. In the fifth case, a 47-year-old woman, the second electrode became dislocated after 2 months. A third attempt was made but failed because of scar tissue in the mediastinum.

#### TERMINATED ATRIAL TRIGGERED CARDIAC PACING

Atrial triggered cardiac pacing was terminated in 17 cases for the various reasons shown in Table 4.

Table 4 *Different causes of termination of atrial triggered cardiac pacing*

Total No.	Inefficient P wave	Unstable P wave	Sinus bradycardia or arrest	Atrial flutter or fibrillation	Other causes see text
17	4	3	4	2	4

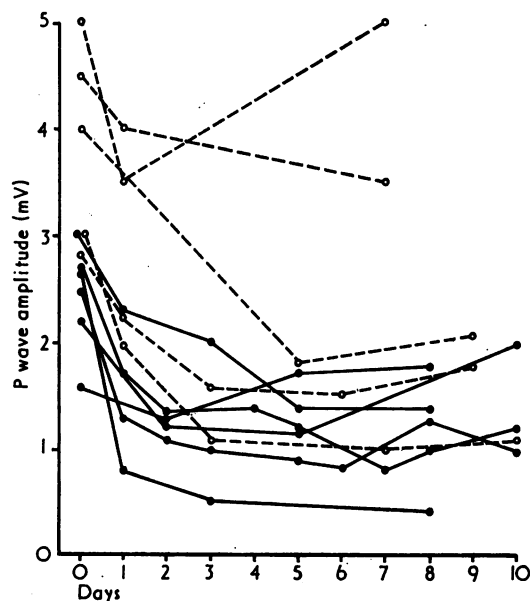


Fig. 5 *P wave amplitude in 11 cases illustrating the typical decrease of the amplitude during the first 10 days; men = ○, women = ●.*

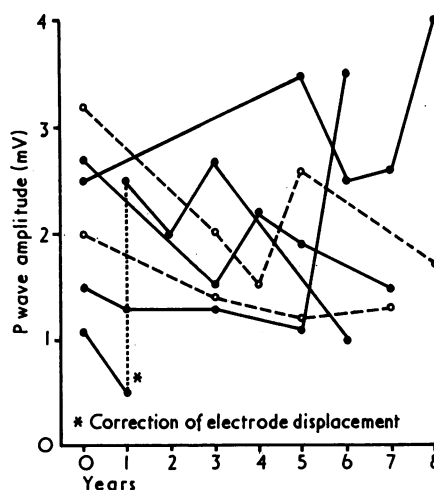


Fig. 6 *P wave amplitude in 6 cases illustrating the variation of the amplitude during treatment; men = ○, women = ●.*

Table 3 *Follow-up of primary P wave*

Acceptable at operation	Inefficient before generator implant	Displacement before generator implant	Inefficient after starting atrial triggered cardiac pacing	Displacement after starting atrial triggered cardiac pacing
No.	No.	No.	No.	No.
Men 44	3	2	4†	3
Women 36	4*	2	3	3
Total 80	7	4	7	6

\*1 patient without x-ray control of the electrode position.

†1 patient with a broken cable.

The 4 cases (3 men and 1 woman) under the heading of 'other causes' include 2 of the men who developed other incapacitating illnesses during atrial triggered cardiac pacing.

The third man had developed atrioventricular block because of myocarditis, and during replacement of a pulse generator two years later it became apparent that the atrioventricular conduction was normal. He was, therefore, given ventricular triggered pacing in order to observe more easily if he could manage without cardiac pacing: this later proved to be the case.

In the case of the woman, recurrent perforations of the cable through the skin of the jugular fossa occurred, accompanied by a slight infection which did not heal until the mediastinal atrial electrode was removed.

In 3 patients, aged 70 to 80, atrial triggered cardiac pacing was temporarily interrupted and changed to ventricular synchronised pacing. In 2 cases this was done because of varying P waves, which were partly inefficient. All 3 patients, however, felt pronounced discomfort with breathlessness and decreased physical fitness, and the cardiac pacing was again changed to atrial triggered cardiac pacing. In case 3 a pulse generator with higher sensitivity to the P waves functioned well.

## Discussion

With regard to the *capability of recording an adequate P wave*, the mediastinal atrial electrode has shown good results initially, with an effective P wave in 80 out of 82 cases. A *preoperative* screening of the mediastinal P wave should preferably be done by an oesophageal electrode to exclude patients with P waves unsuitable for a mediastinal atrial electrode.

The stability of the sinus rhythm should be checked by repeated electrocardiograms including one during exercise. An exercise test with increasing rate of the external pulse generator should also be done to eliminate patients who get anginal pains with increasing heart rate. Some of the early patients in this case material are not representative of patients selected for atrial triggered cardiac pacing more recently. In the early group it was started because of interference between fixed rate pacing and transmitted beats and because atrial triggered cardiac pacing was the first available method of synchronised pacing in this hospital. This explains why it was necessary to replace it with ventricular synchronised pacing in some of the early patients in this study.

The *surgical procedure* gave very little stress to the patients, and was free from complications with the

exception of an easily corrected perforation of the electrode into the pericardium, which was seen in 2 patients. This might have been caused by excessive force on the suction tube.

The atrial fibrillation observed in one patient after implantation of mediastinal atrial electrode continued even after extraction of the electrode. It is possible, however, that the mechanical irritation may have provoked atrial fibrillation in a pre-disposed patient. The atrial fibrillation and flutter observed in 2 patients later on during atrial triggered cardiac pacing seems to be coincidental and an unpredictable cause of terminating it.

The regular decrease of the P wave after operation is most probably caused by tissue reaction against the electrode. During this period ineffectiveness of the P wave without visible displacement on x-ray film occurred only twice. The cause may have been extreme tissue reaction or minor displacement of the electrode. Visible displacement of the mediastinal atrial electrode with an ineffective P wave occurred in 10 patients up to 4 years after starting atrial triggered cardiac pacing. In some patients the cause was obviously an extreme traction of the mediastinal atrial electrode, as described above. After one week the P wave was usually stable during the years of observation. Several mediastinal atrial electrodes are still functioning after 10 years.

The mediastinal atrial electrode has previously been described as a promising method for atrial triggered cardiac pacing (Carlens *et al.*, 1965; Lagergren *et al.*, 1966a, b; Larsson *et al.*, 1969; Larsson, 1970; Kleinert and Nahrstedt, 1974). Larsson *et al.* (1969) presented results similar to this study, with no surgical complication and a displacement frequency of about 10 per cent.

The problem of displacement occurs with other types of electrodes, and our experience shows the importance of securing the electrode and of avoiding traction of it. For practical reasons and to reduce the risk of infection, Larsson *et al.* (1969) have suggested that it is preferable to do one stage implantation of the mediastinal atrial electrode and the atrial triggered pulse generator, even if early displacement of the electrode can still happen.

Apart from the mediastinal atrial electrode (Carlens *et al.*, 1965; Lagergren *et al.*, 1966a, b; Larsson *et al.*, 1969; Larsson, 1970; Kleinert and Nahrstedt, 1974) there are two main approaches for introducing the electrode: implantation via thoracotomy (Center *et al.*, 1963, 1964; Nathan *et al.*, 1964; Center and Nathan, 1966; Silverman *et al.*, 1968; Nusbaum and Levit, 1970; Cleveland *et al.*, 1972; Mills and Ochsner, 1973; Perryman and Sealy, 1973) and transvenous techniques (Rodewald *et al.*, 1964; Center and Castillo, 1967; Center

*et al.*, 1967; Hunt and Sloman, 1968; Kastor *et al.*, 1969; Moss *et al.*, 1969; Kramer and Moss, 1970a, b; Kitamura *et al.*, 1971; Vogel *et al.*, 1971; Rosenkranz and Schaldach, 1971; Bleifeld *et al.*, 1971; Smyth *et al.*, 1971a, b; Porstmann *et al.*, 1972; Zucker *et al.*, 1973; Cappelen and Hall, 1974; Moss *et al.*, 1974; Obel and Scott Millar, 1975; Rubenfire, 1975). Reviews of different methods have been given by Dodinot *et al.* (1969), Sowton (1969), Benchimol and Goldstein (1969), Nathan *et al.* (1971), DeSanctis (1971), and Furman (1973).

The first method described and still most widely used is implantation *via thoracotomy*. With this method the electrode can be fixed directly on the atrium to ensure stability of the electrode. However, the method involves a relatively high risk, as stressed by Dodinot *et al.* (1969). In order to avoid such a major operation other surgical routes to the epicardium have been developed. The retrosternal approach through a subxiphoid incision (Behrends *et al.*, 1965; Bruck and Spang, 1966) seems to offer no significant advantages over thoracotomy.

The problem of the transvenous atrial electrode is the high frequency of electrode displacements (DeSanctis, 1971; Zucker *et al.*, 1973). However, Bleifeld *et al.* (1971), Rosenkranz and Schaldach (1971), and Vogel *et al.* (1971) have reported series of operations almost free from complications. Their methods involved using an electrode held tightly to the atrial wall by barbs or hooks on the electrode tip.

Among other transvenous methods, the J-shaped wires described and used by Kastor *et al.* (1969) and Smyth *et al.* (1971a, b) may be handled and inserted easily but the displacement frequency is high. Zucker *et al.* (1973) described a moderately successful modification of this method. Placing the transvenous electrode in the coronary vein (Hunt and Sloman, 1968; Moss *et al.*, 1969; Kramer and Moss, 1970a, b; Kitamura *et al.*, 1971; Moss *et al.*, 1974; Obel and Scott Millar, 1975; Rubenfire, 1975) has been used for long-term pacing. The potential risk of thrombosis and perforation must be considered but, as far as we know, only one case with perforation has been reported (Kitamura *et al.*, 1971): this occurred with a temporary electrode used in addition for infusion. The experience of this technique is still limited but promising results with low thresholds at generator replacements are reported by Moss *et al.* (1974).

In conclusion, the mediastinal atrial electrode has shown results that are comparable with the best results discussed above and the method has been shown to be suitable for long-term treatment.

The mediastinal atrial electrode has often been

described as involving too high a risk and as being too difficult for general use, but our experience has shown that the method is technically simple and places little stress on the patient. It is, therefore, recommended as a good alternative for P wave detection.

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